

HISTOLOGICAL AND PATHOLOGICAL TECHNIQUES  
UTILIZED IN THE DIAGNOSIS OF CERTAIN  
ANIMAL DISEASES INDIGENOUS TO NIGERIA

by

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## INTRODUCTION

Animal diseases have occurred since very early times and several mentions were made of them in the Bible. They have played a very significant role in the shaping of history as well as the economy of many states and countries. Nigeria is no different in this respect, as certain areas of this African country, particularly the Northern Region, rely heavily on the livestock industry as a pillar of the economic structure.

Certain diseases occurring in Nigeria are indigenous to this geographical area. That is to say, they are present naturally in this country and are not considered as foreign or exotic. Some of the disease problems which plague Nigeria appear also in the United States, with varying frequency, and of necessity, out of economic importance, were included in this report.

Significant publications were present on most of the diseases, but were located in many different references and recorded at different periods in time. It was the intent then, to group together diagnostic procedures concerning these conditions to provide an insight into some of the disease problems, in view of the more recent publications, which may assist in the orientation of veterinarians and animal husbandrymen contemplating a tour of duty in Nigeria.

## ANTHRAX

Anthrax is a disease of most domestic animals and man that has been recorded with the history of man. This disease condi-

tion has been called by other names such as Charbon, Milzbrand and splenic fever. These terms originated from the characteristic lesions noted at the time of necropsy, and were included in many discussions of the disease. (Blood and Henderson, 1960; Henning, 1956; Marsh, 1958; Udall, 1954; Merchant and Packer, 1958; Fincher, et. al., 1956; Siegmund, et. al., 1956; Todd, et. al., 1964)

The causal agent of anthrax is the bacterium, Bacillus anthracis. The organism is an aerobic, capsule producing, gram-positive, spore-forming rod which measures 1 to 1.2 microns in diameter and 3 to 8 microns in length. The spores do not form in the animal body, but necessitate exposure of the vegetative form of the organism to air for this phenomenon to occur. When present, spores were centrally located, ellipsoidal in shape, and measured 0.7 to 0.8 microns by 1.5 microns. Although the general staining characteristics are primarily gram-positive, this reaction is seen in the younger organisms, with a decrease in coloration noted in the older bacteria. The organism tended to form clumps or chains, and appeared flattened at the ends when viewed microscopically.

B. anthracis grew readily on meat infusion agar, and was, in most instances, easily isolated from the blood and internal organs of an infected animal. An alkaline media, pH 7.5-7.8, and incubation temperatures of around 37 degrees centigrade were most conducive to good growth. The colonies appeared dull, opaque, grayish white, and presented an irregular border when grown on an agar surface. If this border were inspected more closely, it

would have been noted to consist of chains of bacteria arranged in parallel fashion, producing the so-called "medussa-head" growth so typical of this organism. When grown in a gelatin stab culture, the growth resembled an inverted fir tree. (Merchant and Packer, 1958)

The organism in the vegetative form was no more resistant than similar cells of other bacteria. However, once exposed to air there was the formation of spores which enabled the cell to withstand adverse environmental conditions. Sporulated forms have been shown to exist and maintain their virulence for periods of 12 years in a buried carcass and 50 years in the laboratory. (Blood and Henderson, 1960; Marsh, 1958; Smith and Jones, 1957; Merchant and Packer, 1958; Fincher, et. al., 1956; Siegmund, et. al., 1965; Todd, et. al., 1964)

Bacillus anthracis is pathogenic for cattle, sheep, horses, mules, dogs, swine, cats and man. The pig appeared to demonstrate the greatest ability to survive the infection, developing primarily an acute pharyngitis typified by extensive collections of edema and hemorrhage. (Blood and Henderson, 1960) The disease infrequently infected the dog and cat, but when present, resembled the disease in swine with the addition of a concurrent gastroenteritis. The most commonly involved animals were cattle and sheep. In these animals the disease appeared to assume at least three forms, viz., peracute, acute and subacute, and was characterized as a fulminating, overwhelming septicemia. (Smith and Jones, 1957) In the first two types, signs were seldom noted in the affected

animals that would lead one to suspect an infection, but were found dead. The subacute infection was typified by diarrhea, fever, subcutaneous collections of hemorrhage and edema, abortion, and death occurred 12 to 36 hours after the onset of symptoms. (Jubb and Kennedy, 1963) Frequent findings in the dead animals included the presence of blood, usually frothy, at the body orifices, and the absence of rigor mortis. (Henning, 1956; Smith and Jones, 1957; Merchant and Packer, 1958; Siegmund, et. al., 1965; Jubb and Kennedy, 1963)

Examination of blood from an infected animal may disclose the presence of numerous gram-positive rods. If antibiotic therapy has been utilized, however, the organisms may be present in numbers insufficient to allow detection by this method. (Jubb and Kennedy, 1963) One author, (Thomson, 1955), suggested in cases of this nature, or in suspected field cases that are microscopically negative for other reasons, that culturing a blood swab in defibrinated blood for approximately 12 hours will frequently allow demonstration of the organisms. The method reported was successful in the diagnosis of anthrax on a par with guinea pig inoculation. The technique described included placing the suspect specimen, consisting of blood, or a swab from that specimen, in approximately 2 ml. of defibrinated blood and incubating this sample at 37 degrees centigrade. Following a minimum incubation period of 12 hours in the media, smears were made from the sample, air dried, fixed by heat and stained with a one percent, aqueous solution of methylene blue. In the positive cases, capsule formation was

generally good and quite often extremely marked. Further, a small number of trials utilizing known anthrax-like organisms failed to demonstrate capsule formation with any of the bacterial strains in defibrinated blood from a variety of animal donors. Other advantages of this technique, when compared with guinea pig inoculation, were that it was cheap, simple, and did not suffer from the occasional death of the guinea pig through the presence of other pathogens.

Regardless of the success one had with either of the above listed methods, it was the consensus of a number of authors that animal inoculation should be performed in all suspect cases. (Smith and Jones, 1957; Merchant and Packer, 1958; Siegmund, et. al., 1965; Williams, et. al., 1965; Jubb and Kennedy, 1963; Coles, 1967) The technique of animal inoculation consisted of either dermal scarification or subcutaneous inoculation. Following death, the anthrax organism could be isolated from the typical cutaneous swellings, heart blood, or spleen of the guinea pig. (Jubb and Kennedy, 1963)

Due to the presence of several non-pathogenic, spore-forming bacilli which resemble B. anthracis, both morphologically and culturally, some investigators believed that it was necessary to differentiate between these non-pathogens and the anthrax organism. (Williams, et. al., 1965) One method for this differentiation is shown in the table below, utilizing differential cultural media and the saprophyte, B. cereus, the non-pathogen most similar to B. anthracis. The table was modified from Williams, et. al., 1965.



Table 1. Cultural reactions of Bacillus anthracis and B. cereus

Cultural condition	<u>B. anthracis</u>	<u>B. cereus</u>
Hemolysis on sheep blood agar	None	Present
Test for mobility on agar	Negative	Positive
Growth in trypticase soy agar	Negative	Positive
Growth on penicillin agar	None	Present

Penicillin agar was prepared by dissolving crystalline penicillin in distilled water, sterilizing the solution by filtration, and adding sufficient material to the melted agar to achieve a concentration of 10 units of penicillin per ml. of agar. Other commercially prepared media were utilized for the remaining characteristics.

Phage typing of suspect cultures has proven beneficial in achieving a definitive diagnosis of anthrax. (Elliott, et. al., 1959) The technique utilized an agar culture of the suspect organism onto which was added one drop of the anthrax specific phage. The phage location was marked with a glass marking pencil. The plate prepared in this manner was then incubated for 18 to 24 hours at 37 degrees centigrade. If the organism in question proved to be B. anthracis, there was an absence of growth in the phage covered area, while growth on the remainder of the plate was unaltered. It has been reported that in some cases there was a rather minimal amount of growth noted in the phage covered area which was believed to be the result of mutant strains or contaminating organisms. Results such as this should not be considered a negative test. No proven strain of B. anthracis has failed to show lysis with the Cherry bacteriophage, and no other bacterial species have



demonstrated lysis in its presence.

Another method described as the "string of pearls" test, has been mentioned and is considered to be beneficial in the diagnosis of anthrax. (Coles, 1967) A 24 hour broth culture of the suspect organism was streaked onto an agar plate containing 0.05 to 0.5 units of penicillin G per ml. of agar. Following a 3 to 6 hour incubation at 37 degrees centigrade, cover slips were placed over the bacterial growth. These areas were then examined with the oil immersion objective. If B. anthracis was present, there were chains of spheres noted which resembled a string of beads. Other organisms form strands or chains of rods. (Coles, 1967)

The gross changes in fatal cases of anthrax appeared to involve primarily the serous tissues of the body and could be termed a polyserositis. (Smith and Jones, 1957) The lesions observed in these locations consisted of hemorrhagic and edematous alterations. Splenomegaly was frequent and apparently a direct result of accumulations of large quantities of unclotted blood. This large volume of blood present in the spleen produced the "currant jelly" consistency, in this organ, so typical of this disease. (Smith and Jones, 1957; Merchant and Packer, 1958; Siegmund, et. al., 1965; Jubb and Kennedy, 1963) Smears and sections of the spleen may reveal very large numbers of the organisms if the carcass is fresh, but when decomposition is advanced the bacilli are destroyed by putrefactive changes. (Jubb and Kennedy, 1963) There was a general lymphadenopathy characterized by collections of edema and occasionally hemorrhage. In cases of long standing, the lymph nodes

became quite enlarged, firm and showed yellowish foci surrounded by connective tissue. (Henning, 1956; Marsh, 1958; Udall, 1954; Smith and Jones, 1957; Fincher, et. al., 1956; Siegmund, et. al., 1965; Jubb and Kennedy, 1963)

Microscopic findings in generalized cases were dominated by the presence of large numbers of anthrax bacilli, except in those instances mentioned previously. The organisms were present not only in the blood, but in other tissues as well. The normal architecture of the spleen was obliterated by the enormous numbers of erythrocytes present. The lymphoid follicles, of this organ if present at all, were very difficult to define. The trabecular remnants of these follicles were the only fact that lent credence to the idea that the follicles did, in fact, exist. (Smith and Jones, 1957; Jubb and Kennedy, 1963)

#### CONTAGIOUS BOVINE PLEUROPNEUMONIA (CBPP)

This disease has been known for some 200 years, and has been responsible for great economic losses both in the United States and abroad. About 1854, the condition was introduced into Africa from Holland where it was eventually responsible for the death of large numbers of cattle. It has remained a very important disease in Africa, ranking second to rinderpest in economical losses. (Smith and Jones, 1957; Todd, et. al., 1964)

The causal agent of CBPP, Mycoplasma mycoides, is a pleomorphic organism which at certain levels of development was noted to be filterable and at others to be visible with the light

microscope. Numbers of synonyms appeared for the organism and included Asterococcus mycoides, Mycoplasma peripneumoniae and Borrelomyces peripneumoniae.

The organism possessed a gram-negative reaction which increased in intensity when Giemsa's staining method was employed. Large masses of these deeply staining spirochete-like organisms may be noted on examination of tissue sections, provided that Zenker's fluid was employed for fixation as formalin readily destroyed the organisms. (Merchant and Packer, 1958)

M. mycoides grows readily at 37-38 degrees centigrade and is fairly sensitive to alterations of incubation temperatures beyond these rather narrow limitations. When incubated for six to seven days at optimum temperature, the virulence of this organism rapidly diminished. These characteristics make the transmission of the disease by fomites a remote possibility at best. (Merchant and Packer, 1958; Huddart, 1960; Turner, 1954)

Clinical signs were initiated by a hacking, dry cough and thoracic pain, noted as occurring during periods of activity, and resembling the symptomatology of a non-specific pneumonia. The frequency of coughing and the severity of thoracic pain increased until marked dyspnea, reluctance to move and grunting during the act of respiration were the prominent signs. (Blood and Henderson, 1960; Henning, 1956; Fincher, et. al., 1956; Todd, et. al., 1964; Hill, 1956) In the later stages, during extensive pulmonary involvement, the animals had mucopurulent exudation at the nares and edematous infiltration of the ventral thoracic region. The

thoracic pain increased in severity until the animal refused to move and the limbs were circumducted to prevent contact between the elbow and the thorax. (Smith and Jones, 1957; Hill, 1956) Following the acute phase, the animals may lapse into semi-acute or chronic form of the condition which, following a course of three to four weeks, may lead to the establishment of a carrier state. (Blood and Henderson, 1960; Henning, 1956; Todd, et. al., 1964; Huddart, 1960; Turner, 1954; Hill, 1956)

The carrier animal made the eradication of CBPP very difficult since no signs were noted to indicate infection. If a carrier animal is stressed by travel or other means, following apparent recovery, the condition may exacerbate and the animal once more begins shedding infective organisms. (Blood and Henderson, 1960; Henning, 1956; Turner, 1954; Hill, 1956)

Transmission of CBPP is by direct inhalation of infected droplets. Due to this mode of transmission, outbreaks are noted with greater frequency in housed animals or in those herded together to allow vaccination or other husbandry practices. The infective focus is frequently provided by a recovered "carrier" animal, in which a pulmonary sequestrum provides a source of infective organisms for prolonged periods of time. (Blood and Henderson, 1960)

The factors which influence the spread of this disease have been well elucidated. (Turner, 1954) These factors included: 1) narrow host specificity of the infectious agent, affecting only bovines; 2) contamination of the environment may be precluded

as contributing to the spread of the disease as the organism is readily susceptible to dessication; 3) susceptible animals must come in close contact with the infected animal to transmit the infection; 4) the most important single factor in the spread of the disease is the inapparent carrier animal; and 5) the long incubation, reportedly to forty days.

Complement fixation is the general standard test procedure utilized for the diagnosis of CBPP. In a portion of the animals, however, the results may be deceptive in that some acute cases presented a negative response, while some positive reactions presented no gross lesions at necropsy. Even though there appeared to be these types of conflicting test results, the complement fixation test remains the test of choice for discovering the carrier state of the disease. (Blood and Henderson, 1960; Henning, 1956; Siegmund, et. al., 1965; Huddart, 1960; Turner, 1954; Hill, 1956)

A modification of the complement fixation test recorded by Huddart, (1960) while on an eradication team in Kenya, seemed to be quite effective. Huddart's technique consisted of removing a small measured amount of blood from the test animal and placing it immediately into an agglutination tube containing saline to achieve a 1:10 dilution of whole blood. The samples were then well mixed and placed in a water bath at 56 degrees centigrade for 30 minutes, followed by centrifugation. The resulting clear supernatant fluid replaced the inclusion of serum in the conventional test procedure. Testing was carried out in plastic

agglutination trays which were incubated by floating on a water bath maintained at 37 degrees centigrade.

Due to the rapidity of Huddart's modification, test results on a herd basis could be expected in about three hours. This short time interval, coupled with the efficiency of the results provided a means to: 1) identify infected herds; 2) provide criteria for early segregation of infected animals in the face of an outbreak; 3) identify and remove from the herd, carrier animals at the close of an outbreak in order to shorten the course of the disease and to lessen the time a herd would have to endure quarantine practices. (Huddart, 1960) The author would hasten to add that one obvious ramification of utilizing such a test for these ends, is that it could provide a means whereby more animals in a given herd could become involved in an outbreak; enhanced by congregating the animals into close quarters to test them.

An interface precipitin test has been described which detected the presence of CBPP antigen. This technique seems to have more utility as a diagnostic evaluation of the lesions discovered at necropsy rather than detection of infected animals. Turner (1954) prepared the specimen by finely grinding one gram of the suspected lesion and suspending the sample in 10 ml of saline. This suspension was then shaken vigorously by hand with an equal volume of chloroform to extract the lipid fraction. This procedure was repeated until the supernatant fluid was clear. On occasion, it proved necessary to acidify the saline suspension (pH 5) or submerge the specimen into boiling water to precipitate the protein prior to



the chloroform extraction in order to provide a clarified supernatant solution. Serum (antibody source) for the test was collected from a virtually non-clinical case of the disease seven days preceding the first positive complement fixation reaction. The serum collected in this manner was placed in the bottom of a Dreyer tube and the antigenic material extracted from the lesion was carefully layered over the serum. Strong reactions were noted in approximately a minute or so, but tests were allowed to continue for some 30 minutes before being ruled negative.

The test procedure described proved of value in detecting a high percentage of both acute and chronic lesions. The antigen was very resistant, typified by recorded positive reactions in formalin preserved museum specimens believed to be 24 years old. It also resisted destruction by putrefactive processes, since positive reactions were obtained in some instances with this type of material up to 21 days following placement of the antigen in a putrefactive exudate. The test for antigen did not compare favorably with the complement fixation test, however, since it gave negative results while complement fixation remained positive. (Turner, 1954)

The lesions of CBPP, as one might expect, were primarily confined to the respiratory system and most striking. There were noted, in most cases, lesions consisting of thickened pleura accompanied by heavy fibrinous depositions, a fibrinopleuritis. There was also an increase of blood-tinged serous fluid in the pleural cavity. Areas of the lungs showed interlobular edema,



marked regions of pleural adhesions, and inflammation of bronchiolar membranes. The lungs experienced various stages of hepatization, from red to grey, producing the characteristic "marbling" so frequently associated with this disease. (Smith and Jones, 1957; Todd, et. al., 1964) In the more chronic or carrier form, there may be necrotic pulmonary lesions surrounded by dense regions of connective tissue which formed the sequestered region identified with the carrier animal or the so-called "lunger" syndrome.

The microscopic lesions consisted predominantly of separation of lung lobules by distinct areas of connective tissue infiltration. There may be normal alveoli noted within some lobules, while others were in an advanced state of hepatization and quite well consolidated. Lymphocytic infiltration was marked perivascularly and around the periphery of the bronchi. Leukocytes, predominantly neutrophils, were the most common infiltrating cell type in the interlobular septa. (Smith and Jones, 1957; Siegmund, et. al., 1965; Todd, et. al., 1964; Jubb and Kennedy, 1963; Hill, 1956)

#### CUTANEOUS STREPTOTHRICOSIS

This condition, a mycotic dermatitis, was found to occur in all species, but was of economic importance primarily, only in cattle and sheep. It has been suggested to refer to the condition in cattle as cutaneous streptothricosis and in sheep as mycotic dermatitis. (Blood and Henderson, 1960) The causal agent of this syndrome was the actinomycete, Dermatophilus congolense. Early

names for this condition, which are noted as synonyms today, included contagious dermatosis, impetigo, dermatomycosis and bovine tropical impetigo. (Plowright, 1958) Although large death losses are not recorded with this disease it achieves its economic importance by producing damage to the hides and affecting a weight loss on severely infected animals.

This disease has almost universal occurrence in Africa, particularly in the regions located south of the Sahara. It appeared to occur with seasonal frequency in these areas, primarily during the periods of prolonged rainfall. (Plowright, 1958; Szabuniewicz, 1964)

Clinically, the observable alterations appeared to be divided into three main phases. Initially there was an exudative dermatitis characterized by multiple slightly elevated areas in which the ends of the hair were matted together and standing erect, resembling a paint brush. The matted hair could easily be removed from the area with only slight traction, leaving a rather convex lesion covered with a purulent exudate.

Following these earlier changes, the exudate appeared to coalesce forming dense, scab-like lesions which were grey in color and firmly attached to the skin. The lesions gradually became firmer and increased in size to form "horny" projections, particularly over the shoulders. The lesions at this time were similar to those noted in other hyperkeratotic conditions.

Finally the large scabs sloughed leaving normal skin. New hair growth will be noted as clinical recovery occurs.

(Szabuniewicz, 1964)

There appeared to be no pruritis associated with the lesions in any of the cattle observed. The crusts formed were characteristically thick and measured some one to two inches in diameter, and had a rather uniform distribution over the animal body.

As a rule the general health of the animal was not affected unless the lesions were widespread or they became secondarily infected. In these instances, the animals demonstrated poor conditioning and frequent deaths were noted. There were reports concerning the death of an occasional animal from pneumonia caused by infection with D. congolense. (Blood and Henderson, 1960)

Transmission appeared to stem from contact with infected animals or contaminated insect vectors. The etiological agent has been transmitted under experimental conditions by the stable fly, Stomoxys calcitrans, and the common house fly, Musca domestica. It was demonstrated that mechanical disruption of the skin was not necessary for the transmission to occur, but when present it assisted in the dissemination of the organism. It was also evident that moisture applied to both the donor and recipient animals enhanced transmission of this condition. (Richard and Pier, 1966)

Other agents believed involved in the transmission of streptothricosis are insects such as the tick, Amblyomma variegatum, debilitating factors such as trypanosomiasis, parasitism, undernourishment, etc., and injuries to the skin such as those created by tick birds and barbed wire. (Plowright, 1958)

The gross necropsy lesions were rather meager, confined to the skin, and resembled the clinical description. In the occasional

animal death from pulmonary infection, the lesions resembled those of a secondary pneumonia. (Blood and Henderson, 1960)

Histopathological lesions were likewise confined to the skin with the hyphae of the organism penetrating the stratum corneum and extending within it to form envelopes of coccoid spores. The reaction in the epidermis which resulted in inundating the region with an exudate composed of erythrocytes, leukocytes, and highly keratinized epithelial cells. Many micro-abscesses and necrotic foci were commonly noted, but the most outstanding change was the separation of the stratum corneum into layers by the focal accumulation of neutrophils, serous exudate or erythrocytes. (Plowright, 1958; Szabuniewicz, 1964; Pier, et. al., 1963)

A presumptive diagnosis may be achieved by several methods. Five percent bovine flood agar has been utilized for primary isolation of the organism with detectable growth reported in 24 to 48 hours. Several stains including methylene blue, Giemsa and carbol thionin were utilized for microscopic examination of the organism, however Gram's stain proved to be superior to any of the others on trial. (Plowright, 1958)

A hot five percent solution of sodium or potassium hydroxide has been employed into which the "crusty" lesions were placed for several minutes. Following the clearing action of the hydroxide solution, the solid material remaining in the tube was spread in a thin film on a microscopic slide, fixed by heat or alcohol, stained with Giemsa's stain for 30 minutes and examined microscopically for the hyphae associated with this organism. (Szabuniewicz, 1964)

From the work of Kelley, et. al., (1964) a much simpler technique was described which consisted of rubbing the concave surface of the lesion directly on a microscopic slide, fixation by methyl alcohol, and staining with Giemsa's stain. This technic disclosed the gram-positive filaments and spores characteristic of this organism. (Richard and Pier, 1966; Plowright, 1958; Szabuniewicz, 1964; Pier, et. al., 1963; Kelley, et. al., 1964)

Positive identification of the organism was performed utilizing pulverized scab material and its inoculation into blood agar plates containing 1,000 units of Polymixin B per ml. of media. (Kelley, et. al., 1964) Since the growth of D. congolense was inhibited by bacterial contamination, addition of this antibiotic appeared beneficial in the isolation of this agent. (Plowright, 1958; Szabuniewicz, 1964; Pier, et. al., 1963) Growth on blood agar was accompanied by slight beta hemolysis with some of the growth, yellow in color, occurring beneath the agar surface. (Kelley, et. al., 1964)

It has been demonstrated that an infection with D. congolense produces a detectable antibody titer. (Pulliam, et. al., 1967) The titer of the animals on test, naturally and experimentally infected, was of sufficient magnitude to detect by several immunological procedures which included agglutination, agar gel precipitation, and indirect hemagglutination (IHA) tests.

Serum for the test procedures was prepared by injecting a standardized quantity of sonified cocci and spores taken from cultures of D. congolense. The animals received 1 subcutaneous and 1 intradermal inoculation each week for a period of 4 weeks.

Following this period of hyperimmunization the animals were bled and the test serum prepared.

Animals hyperimmunized in this manner had agglutination titers of 1:160 to 1:640, positive gel precepitation and IHA titers of 1:80 to 1:2,560. Test animals had an equally strong pattern with agglutination titers of 1:80 to 1:640, positive gel precipitation, and IHA titers of 1:80 to 1:2,560. At the same time, normal controls had titers with agglutination and IHA of up to 1:40 and negative gel precipitation.

It would seem from the foregoing data that immunological testing is capable of a strong contribution toward the definitive diagnosis of streptothricosis.

#### RINDERPEST

Rinderpest is a contagious, highly fatal disease of cattle characterized by necrotic stomatitis and gastroenteritis. This condition has been described since ancient times as one of the most devastating animal diseases. (Blood and Henderson, 1960; Scott, 1964) It has been stated, "that rinderpest still had more influence on the world's food supply than any other animal disease and as late as 1949 was responsible for over two million cattle deaths annually". (Todd, et. al., 1964)

The etiological agent of this disease is a virus which demonstrates an affinity for the epithelium of the gastrointestinal and respiratory systems. (Blood and Henderson, 1960; Todd, et. al., 1964) The agent also appeared to be attached to the leuko-



cytic elements of the blood, thus accounting for the non-pathogenic effect of serum or erythrocytes which have been injected into susceptible animals. (Scott, 1964)

There are apparently many different strains of the virus, differing from one another in pathogenicity, ease of transmission and host affinity, but being immunologically identical. (Blood and Henderson, 1960; Todd, et. al., 1964; Scott, 1964) The agent is very fragile, being unable to survive outside the host or in a cadaver for any extended period of time. (Blood and Henderson, 1960; Scott, 1964) The virus has been provisionally classified with the myxoviruses and ranges in size from 120-300 mu. (Todd, et. al., 1964; Scott, 1964)

Rinderpest was usually spread by direct contact between susceptible and infected animals. It is believed that virulent virus particles are shed in the nasal and lacrimal secretions which are capable of contaminating food, water and other areas of the environment. The wild-life population, when infected, may harbor the infection and continue to disseminate it for many years. This latter fact made the eradication not only difficult, but in some areas impossible. (Blood and Henderson, 1960; Todd, et. al., 1964; Henning, 1956)

Clinically the symptoms are complicated by the differences in strain virulence which are capable of producing infections over a wide range of clinical severity varying from inapparent to per acute. Following natural infection and an incubation period ranging from 3 to 15 days the first symptom was an abrupt elevation in temperature to 104 to 105 degrees Fahrenheit.



Nasal and lacrimal discharges, usually serous in nature, soon followed with concurrent anorexia, depression and polydipsia. The serous discharge later became mucopurulent in nature with vascular injection detectable in all observable mucous membranes.

Following 3 to 5 days of fever, small pin point vesicles were noted on the oral mucosa. These areas appeared to enlarge, coalesce, and finally necrose, producing hyperemic erosions accompanied by excessive salivation.

Diarrhea, frequently hemorrhagic in nature, was frequently seen as the temperature reached its peak and began to decline. This event was observed on or about day 5. The symptoms associated with the disease at this stage included abdominal pain, emaciation, severe dehydration, accelerated respiration and prostration with death occurring 6 to 12 days after the febrile onset. (Blood and Henderson, 1960; Smith and Jones, 1957; Todd, et. al. 1964; Scott 1964)

Skin lesions have been described (Scott, 1964; Gillespie, 1966; Beaton, 1966) but their significance has yet to be determined. Some investigators (Scott, 1964; Gillespie, 1966) feel the skin lesions represent a more virulent form of rinderpest while others (Beaton, 1966) believe it to be either a strain of reduced pathogenicity or a suprainfection with streptothricosis.

Gross lesions appeared to be limited primarily to the gastrointestinal tract and the lymphatics. Observation of the lymph nodes may disclose edematous changes due to the direct action of the virus. Peyer's patches were frequently eroded, leaving craters

in the intestinal wall which, in most instances, were hemorrhagic.

In the digestive system the lesions were characteristic of the anatomical region involved. The oral cavity was well marked with hemorrhagic ulcerations. These erosions were rather shallow with a reddened, raw-appearing core and were well delineated by normal epithelium. The lesions appeared to be selectively located, principally on the inside of the lower lip, the adjacent gum, the cheeks at the commissures and the ventral free portion of the tongue. The dorsal, cranial portion of the tongue was very seldom, if ever, involved with these ulcerations. The pharynx and proximal one-third of the esophagus had lesions similar to those of the oral cavity. The rumen, reticulum and omasum were seldom involved.

The abomasum was the most frequently affected organ. The lesions were in abundance in the pyloric region and consisted of irregularly outlined hemorrhagic areas varying in color from bright red to brown. These lesions demonstrated a tendency to follow the plicae from the pylorus cranially to the fundus, decreasing in severity as they progressed. Edema was prominent in the plicae. This resulted in an increase in the thickness of the folds and imparted a gelatinous appearance to the cut section. If the animal succumbed to rinderpest there was frequently no abomasal content, excepting the accumulation of a blood tinged mucous exudate.

The small intestine demonstrated involvement similar to the erosions described in other areas. Peyer's patches frequently were absent having eroded to leave in their place deep, raw craters.

The remaining portions of the digestive system were affected in the same manner as the abomasum, but the severity of these alterations was less marked and considerable variation was noted.

The epithelium of the respiratory system is also susceptible to the action of the virus. Petechiation accounted for the majority of the gross lesions in the area of the turbinates and larynx. The cranial portion of the trachea had lesions consisting of streaks of rust-colored hemorrhage which were marked by the absence of mucosal erosions. In cases of long standing, in which the animal was prostrate, there may be interlobular and alveolar emphysema accompanied by slight hemorrhage and consolidation.

The microscopic lesions are striking, particularly in the lymphoid tissue. The rinderpest virus causes necrosis of the lymphocytes producing massive destruction and disappearance of mature lymphocytes. The lymphocytic elements were replaced by an eosinophilic, acellular matrix. This matrix, as a general finding, was surrounded by macrophages, degenerating lymphocytes, plasma cells and nuclear debris.

The changes in the abomasum were marked by necrotic foci in the epithelium and hemorrhage in the underlying lamina propria. Deep ulceration was noted on occasion and extended to penetrate the muscularis mucosa.

Diagnosis, at least presumptive, may be achieved by taking into account the clinical signs, the gross lesions and the local epizootiological conditions. Confirmation of this tentative diagnosis rests in: 1) challenge of susceptible and immune animals with splenic suspensions collected from the suspect during the

febrile phase; 2) demonstration of antibodies specific for rinderpest by complement fixation; or 3) serum virus neutralization tests in tissue culture. (Henning, 1956; Smith and Jones, 1957; Fincher, et. al., 1956; Todd, et. al., 1964; Scott, 1964)

### FOOT AND MOUTH DISEASE

Foot and mouth disease (FMD) is an acute, extremely contagious, viral disease which occurs naturally in all cloven hooved animals. The disease not only affects cattle, sheep and swine, but in addition the wild ruminants such as deer, goats, and antelope. (Blood and Henderson, 1960; Todd, et. al., 1964) Under some conditions these wild animals served as a reservoir for the infection. (Smith and Jones, 1957) The condition is known by such other names as aphthous fever, epizootic aphtha, and Maul-und Kauenseuche. (Todd, et. al., 1964)

The virus has been shown to exist in seven antigenic strains. Type O appeared with the greatest frequency and type C the lowest. Occurring at points between O and C were types A, SAT-1, SAT-2, SAT-3, Asia-1 and the various subtypes of each. The SAT-1 through SAT-3 types have occurred to date only in Africa. The symptoms and lesions produced by these various viral strains are very similar, but infection or vaccination with one type did not confer immunity against the others. (Smith and Jones, 1957)

The infectious agent has been demonstrated to be spherical in outline and measured 22-23  $\mu$  in diameter. The agent, when free in the environment demonstrated a degree of resistance, but when

protected by mixture with tissue or in feed stuffs it would remain viable for periods up to one year. It was this property that allowed the easy introduction of the disease into previously non-infected countries by transportation of the virus in leather goods, meats and their by-products or by simple contamination of a piece of wearing apparel or vehicle. (Todd, et. al., 1964)

The virus appeared in the blood, milk and in the saliva before the appearance of vesicles in the mouth. All body excretions, including milk, semen, urine and feces were infective before signs of clinical illness became apparent and for a short time proceeding an apparent recovery. Therefore, contact with an infected animal or contaminated environment may transmit FMD to a susceptible animal. (Blood and Henderson, 1960; Henning, 1956; Smith and Jones, 1957; Todd, et. al., 1964; Jubb and Kennedy, 1963)

Clinically, the incubation period of 1 to 21 days may go undetected with the first alteration consisting of vesicular formation in the oral cavity and in the interdigital spaces. If symptomatic, there was an initial febrile period with temperatures as high as 104 to 106 degrees Fahrenheit recorded. This febrile period was accompanied by depression and finally anorexia with the advent of the vesicular formation and subsequent stomatitis. At this particular time in the progression of the disease, the fever began to subside, salivation was marked, and some abortions were noted. The saliva produced was characteristically thick, ropy and tenacious. The animal had a tendency to smack its lips and to exercise a great deal of caution during normal masticatory procedures.

Vesicles, measuring 1 to 2 cm., appeared on the buccal mucosa,

on the dorsal surface of the tongue, and on the dental pad. They usually ruptured in about 24 hours to leave a raw painful region, hemorrhagic in nature, that required 7 to 10 days to heal. The periphery of this lesion was usually irregular in outline due to the remnants of the necrotic epithelium which persisted following rupture of the vesicles. (Todd, et. al., 1964) Concurrently with the oral lesions, vesicles appeared on the feet at the coronary border, in the interdigital clefts, and on the sparsely haired areas of the body such as the teats, between the thighs, etc. (Blood and Henderson, 1960; Henning, 1956; Smith and Jones, 1957; Fincher, et. al., 1956; Todd, et. al., 1964) Rupture of vesicles on the feet was usually accompanied by marked discomfort evidenced by lameness. During this time there was also a painful swelling located at the coronary border. (Blood and Henderson, 1960; Todd, et. al., 1964)

The possibility of a carrier state in FMD exists, since viable viral particles have been demonstrated in the saliva from a significantly large number of cattle as long as 8 months following recovery from the clinical disease. (Sutmoller and Gaggero, 1965)

The gross lesions consisted primarily of the vesicular lesions over the sparsely haired areas, feet and in the oral cavity. In addition to the vesicular lesions, there may be punctate hemorrhages and/or edema of the abomasal and small intestinal mucosae. The mucosa of the large intestine, however, was simply hyperemic and blue-red in color.

Other lesions noted were associated with cardiac and skeletal



muscle. The lesions consisted of linear areas of necrosis and leucocytic infiltration which were greyish-white in color. This typical striping of the myocardium was responsible for some referring to the cardiac lesions as "tiger heart". (Smith and Jones, 1957)

The microscopic picture was concerned primarily with the development of the vesicles, from their inception until rupture, and did not add significantly to the diagnosis.

Differential diagnosis required testing procedures to rule out the possibility of vesicular exanthema and vesicular stomatitis. There were primarily two areas in which clinical pathological testing was employed, namely complement fixation and experimental transmission in susceptible test animals.

Type specific and strain-specific complement fixing antisera are available, which permits strain and type identification of the virus during an outbreak. Diagnostic antisera are also available to distinguish vesicular stomatitis from FMD. (Blood and Henderson, 1960)

Weanling white mice provided a suitable test animal in which to detect viral particles in a suspect specimen. In white mice injected with the virus, an acute myositis was produced which resembled the myositis discovered in infected cattle. (Blood and Henderson, 1960; Smith and Jones, 1957; Todd, et. al., 1964) Guinea pig inoculation has also been utilized in the diagnosis of FMD. The inoculum consisted of vesicular fluid withdrawn from the suspect animal and injected subcutaneously into the plantar pad of the guinea pig. Vesicles appeared on the pads of the



guinea pig 1 to 7 days post-inneculation and in the oral cavity 2 days later in the positive cases. (Blood and Henderson, 1960)

### HEARTWATER

"Heartwater is a septicemic, infectious rickettsial disease of sheep, goats and cattle, but all ruminants may harbor the causative agent, although some ruminant types and species may not show apparent clinical manifestations." (Todd, et. al., 1964) This condition has been variously referred to as heartwater, hartwater, bossiekte, gallsiekte, and drunken, mad or black gall sickness. (Henning, 1956)

The etiological agent is Rickettsia ruminantium or Cowdria ruminantium which demonstrates a predelection for the endothelial lining cells of blood vessels in the mammalian host. In the invertebrate host it appeared confined mainly to the epithelial cells of the intestine and the lumen of the gut. The organism has a coccoid form and measures from 0.2 to 0.5 microns in diameter. In tissue section, these organisms were clumped together with all those present in one area being nearly the same size. There were differences in size noted between the different clusters of the rickettsial bodies, however. (Henning, 1956)

The agent demonstrated very little resistance once removed from the animal body. Infective blood removed from the host became innocuous within a short period of time. (Henning, 1956; Todd, et. al., 1964)

The clinical period of this disease, which followed a variable incubation period of from 14 to 28 days, was characterized

by four forms, namely, peracute, acute, subacute, and mild.

The peracute form primarily involved imported cattle, sheep and goats. (Todd, et. al., 1964) This fact alone would place the disease in an economically important position, particularly in view of the recent cattle importations into Nigeria as a part of U.S.A.I.D. program to upgrade the cattle in that country.

The symptomatology associated with this form of the disease was a fever of 106 to 108 degrees Fahrenheit, convulsions and death. (Todd, et. al., 1964) The disease may be so sudden that an apparently normal calf, when placed with its dam, would commence to nurse energetically, and then suddenly drop to the ground, experience a few convulsive seizures and die. (Henning, 1956)

The acute form of this condition is by far the most frequently encountered of the four types. The initial symptom noted was confined to hyperthermia. The animal seemed to have a normal appetite and ruminations early, but gradually became increasingly inappetent and depressed.

The next observable sequela was involvement of the central nervous system. There was abnormal masticatory movement, protrusion of the tongue, twitching of the eyelids, a rather characteristic high-stepping, unsteady gait, and the animal may stand with legs spread and head pressed hard against an immovable object. Eventually the animal became prostrate and experienced convulsions characterized by galloping movements, extension of the head and neck and frothing at the mouth. (Henning, 1956; Todd, et. al., 1964; Clark, 1962) During this final period, there was an exag-

gerated response to reflex stimulation; even the slightest cutaneous stimulus was capable of evoking the most pronounced nervous symptoms, resembling those of strychnine poisoning. (Henning, 1956; Todd, et. al., 1964)

Signs of the subacute and mild form resembled those of the previous two forms, but differed from them in that they were less severe and more prolonged. Recovery from these types occurs with greater frequency, but the convalescent period was often quite lengthy. (Clark, 1962)

Wild animals were involved, but seldom displayed the symptomatology associated with this disease. These individuals usually recovered in a short period of time from a mild febrile reaction but continued to harbor the rickettsia asymptotically, as was demonstrated by transmission studies. (Henning, 1956) The inapparency and persistence of this latter type of infection created a difficultly diagnosed reservoir for future outbreaks. (Todd, et. al., 1964)

The gross lesions were fairly uniform in all of the animals observed. The picture was typified by ascites, hydrothorax and hydropericardium. The latter lesions occurred with such frequency as to provide the name for this disease.

In addition to these changes there were also scattered areas of hemorrhage, hyperemia and edema located in the lymph nodes, lungs, and spleen.

The mucous membrane of the abomasum, in the region of the pylorus, was swollen, diffusely hyperemic and displayed a number

of hemorrhages over its surface. Similar changes occurred throughout the remainder of the intestine but failed to be as marked. (Henning, 1956; Todd, et. al., 1964; Clark, 1962)

Diagnosis of this disease has been rendered quite difficult due to the rather short termed existence of the etiological agent and its staining properties. Elaborate procedures have been described to accomplish a definitive diagnosis, but due to the complexities involved they are impractical in field diagnosis. (Henning, 1956; Todd, et. al., 1964)

A method has been described which has been utilized with efficiency in field diagnosis. (Purchase, 1962) The technique consisted of removing a small sample, tomato seed sized, from the grey matter of the cerebrum. This material was placed on a glass microscopic slide and another slide applied over the specimen and the first slide. Sufficient pressure was then applied to spread the tissue over the greater part of the width of the slides, the upper slide was raised to an angle of two to five degrees and drawn along the lower one, maintaining moderately firm pressure on the slides the whole time.

The smears were air dried, fixed in methyl alcohol and stained with a 1:10 dilution of Giemsa's stain for thirty minutes. The stained slides were then washed in running tap water for 5 minutes and allowed to dry or were blotted dry.

The scanning lens of the microscope was utilized to locate a field of capillaries. These areas were then carefully studied with the oil immersion lens to locate the dark blue bodies of

R. ruminantium. The rickettsial bodies may be distinguished from the nuclei of endothelial cells, in that the latter stain purple instead of blue, by the technique described above. (Clark, 1962)

Smears made from the intima of the jugular vein and stained in a similar manner have been utilized with results similar to those reported above.

Animal inoculation has been employed, but once again the fragility of the infectious agent dictated the inoculation be accomplished as soon as possible after collection. Perpetuation of the organism by ferret or mouse inoculation has proven beneficial for returning the organism to the laboratory, from a field case, in a viable form. (Todd, et. al., 1964)

#### TRYPANOSOMIASIS

"This term has been applied to a group of acute or chronic infectious diseases in man and animals, due to the invasion of the body by unicellular blood parasites, trypanosomes." (Henning, 1956) The agent produces, among other conditions surra, nagana, sleeping-sickness, and dourine. (Blood and Henderson, 1960; Henning, 1956; Smith and Jones, 1957)

The life cycle of these agents required both a vertebrate and an invertebrate host. Flies of the genus Glossina, Tachinoides, and Palpalis, the so-called tsetse flies, have been shown to harbor and transmit certain trypanosomes. (Godfrey and Kendrick, 1961; Jordan, 1965; Godfrey, et. al., 1965; Ford, 1965) In these invertebrate hosts, the trypanosomes pass through different developmental

stages until the infective stage has been achieved. Once infective, the agent migrates from the gastrointestinal tract to the salivary gland of its host ready to infect the next vertebrate host on which the fly feeds.

Trypanosomes, regardless of species, share certain anatomical features. They have an ovoid or rounded body in the non-flagellate stage and a slender elongate body when flagellated. The cell is surrounded by an undulating membrane which terminates in a variably developed flagellum. A rather prominent nucleus is present near the center of the cell and is marked by the presence of a more deeply staining karyosome. (Smith and Jones, 1957)

Since several trypanosomes are capable of pathogenesis, each condition occurring in Africa will be discussed as to etiology and diagnosis under the general heading of trypanosomiasis.

#### I. Nagana, Tsetse-fly Disease

Nagana is a frequently utilized term which encompasses the African Trypanosomiasis that involve domestic animals, particularly infections with T. vivax, T. brucei, T. congolense. (Smith and Jones, 1957) These organisms may be found singly or concurrently as mixed infections. The severity of the disease produced does not seem to be markedly influenced by the presence of mixed infections. (Godfrey and Kendrick, 1961; Jordan, 1965; Godfrey, et. al., 1965; Malmquist, 1965) The term "Souma" is sometimes used in reference to an infection with T. vivax. (Smith and Jones, 1957) A large variety of native game animals were found to be infected naturally, and to serve as an asymptomatic reservoir of the infection.



T. Congolense was monomorphic and the smallest of the pathogenic trypanosomes, measuring 8 to 21 microns in length and 1 to 2.7 microns in width. The undulating membrane was poorly developed and there was no free flagellum, but in spite of this the organism was fairly active in fresh smears. (Henning, 1956) This was the agent most commonly found infecting Zebu cattle in Nigeria. (Godfrey, et. al., 1965)

T. brucei occurred pleomorphically, thus significantly complicating the description of the microscopic appearance. The pleomorphic forms of this organism may be grouped into three general categories, namely, short, broad non-flagellated types, long, slender flagellated organisms and the intermediate, variably flagellated forms.

Movement, present in fresh preparations of the organism, was characterized as oscillating, with little forward progress noted. Some of the slender forms however, were capable of a minimal amount of forward motion.

T. vivax, the most highly motile of the trypanosomes, had very characteristic morphology. The bulk of the cytoplasm is located behind the nucleus which created an enlarged, piriform caudal extremity. The organism measured 18 to 26 microns and possessed well developed flagella which averaged 6 microns in length. (Henning, 1956)

Although some breeds of cattle in West Africa are immune, most other breeds, whether imported or native, are susceptible to this condition. In susceptible cattle, the disease occurred as either an acute or chronic infection which was manifest by irregular



fever, loss of condition, anemia, weakness, subcutaneous edema and in some animals by a photophobia. (Henning, 1956; Jordan, 1965; Godfrey, et. al., 1965) It was reported that there was a significant decline in serum protein, hemoglobin values, red cell volume, and total erythrocytes during the course of the infection. (Godfrey, et. al., 1965) Death is the expected termination in the untreated individual.

Necropsy lesions were marked by severe emaciation, edematous changes in the adipose tissue and in some cases, a rather anemic cadaver. The liver was firm, enlarged and congested and the cut-surface bulged slightly when incised. The lymph nodes were enlarged from edema and occasional hemorrhages were noted in the medulla. (Henning, 1956; Smith and Jones, 1957) Hemorrhages were found with frequency in the subendocardial and epicardial areas of the heart. (Smith and Jones, 1957; Godfrey and Kendrick, 1961; Godfrey, et. al., 1965) An abomasitis characterized by congestion and hemorrhage was reported, but due to the presence of a concurrent Haemonchiasis in the test animals, the exact etiology of this finding was not established. (Godfrey, et. al., 1965)

Diagnosis is usually based upon finding one of the organisms in a blood smear. The most widely described technique consisted of preparing both thick and thin blood smears from the suspect animal. The thick smears were lysed with distilled water, fixed by methanol, stained with Giemsa's at a dilution of 2 drops of stain to each ml. of water, and were examined under the 40x microscope objective. The purpose of this film was to establish the

presence of the organism. No attempt was made to identify any trypanosome found, due to the amount of optical distortion created by this technique.

The thin blood films were air dried and stained in the same manner as were the thick smears. Films found positive were studied closely to identify the involved trypanosome.

An additional method, which proved more satisfactory than did the blood smears, was animal inoculation employing the white rat. The inoculum consisted of 3 ml. of citrated blood drawn from the suspect animal and injected intraperitoneally.

Blood smears were performed on the rats, in the manner outlined previously, beginning on the fourth post-inoculation day and continuing on a daily basis thereafter until the organism was located or until 10 days had lapsed.

The rat inoculation technique uncovered several cases of trypanosomiasis which had remained sub-patent microscopically. The technique did not, however, recover T. vivax. It was believed to be a more efficient diagnostic method than the microscopic technic in that more of the suspect blood volume was examined and therefore the possibility of finding an infection in the suspect animal should have been greater. (Godfrey and Kendrick, 1961; Godfrey, et. al., 1965)

## II. Dourine, Equine syphilis, Slapsiekte

This is a contagious trypanosomiasis of the equine which was transmitted by coitus and characterized by inflammation of the external genitalia, skin lesions, and paralysis.

The protozoan involved in the etiology of this infection was T. equiperdum. This organism is incapable of surviving either outside the animal body or in cadavers, thus minimizing the spread of infection from contaminated environment. The agent localized in the urethra or in the vagina and was demonstrable in smears made from washings of these areas. The organisms periodically disappeared from these locations so that they could have been easily missed during the routine clinical examination of an animal. There is a further complication in that some animals are capable of being clinically normal carriers of the infection. (Blood and Henderson, 1960; Henning, 1956; Smith and Jones, 1957)

Clinically, the disease is characterized by edema of the external genitalia to the extent that paraphimosis was noted with frequency in the stallion. This edematous change spread rapidly to the perineum and along the ventral abdomen as far forward as the sternum. There was often a muco-purulent urethral discharge in the stallion and a similar secretion from the vagina in mares.

Urticarial-like skin plaques developed, which measured 1 to 2 inches in diameter, and were located over the neck and trunk of the animal. This cutaneous involvement persisted for several weeks.

Nervous signs made their appearance a variable time after the edematous and cutaneous alterations and were due to the specific action of the organism or toxins produced by the organism acting directly on certain nerve cell bodies and their axones. The initial changes consisted of weakness and stiffness of the hind limbs which

was characterized by marked incoordination. This gradually intensified until the animal dragged one or both hind limbs consistently enough to show wear on dorsal aspect of the toe of the hoof. Marked atrophy occurred in the hind quarters and all animals suffered from emaciation, which became so severe in some animals as to necessitate their destruction. Extreme variability was noted in the severity of the clinical picture due to the presence of different strains of the protozoon each exhibiting varying degrees of virulence. (Henning, 1956)

Gross lesions noted at necropsy were similar to the lesions described in the clinical picture.

Diagnosis was made chiefly from the characteristic clinical picture and demonstration of the causal trypanosome in vaginal and urethral washings and in fluid collected from the edematous swellings. In clinical cases involving the more pathogenic varieties of the organism, it was possible to locate the trypanosome in blood smears utilizing the techniques described earlier.

Diagnosis of dourine rests in locating the causal organism in either the blood or washings of the genitalia, the typical clinical syndrome and in some instances the demonstration of complement fixing antibodies. The application of a complement fixation test has proven itself beneficial in the eradication of this condition from Canada. Reagents employed in this test appeared to share reactive properties with T. brucei. (Henning, 1956; Smith and Jones, 1957; Siegmund, et. al., 1965)

### III. Surra

This condition, caused by T. evansi, is widespread in most tropical and subtropical countries. The disease apparently originated in Africa from an infection of camels with T. brucei. The camels were withdrawn from the fly belts along the trade routes to the north through the Sahara, and the organism underwent mutation, sufficiently so as to forego the previously described developmental phases which required an invertebrate host. It then came to depend upon Tabanidae spp. and Stomoxys spp. for mechanical transmission to other susceptible hosts. (Siegmund, et. al., 1965)

T. evansi resembled the long form of T. brucei and measured 24 to 38 microns in length and 1 to 1.5 microns in breadth. As mentioned above, this trypanosome depends on invertebrate transmission, but presumably does not require it for developmental purposes. (Henning, 1956; Siegmund, et. al., 1965)

Tabanidae and Stomoxys have been incriminated in the transmission of this agent as well as leeches, ticks and vampire bats. There have been instances in which the animals were believed to be infected from the ingestion of freshly killed infected meat. (Siegmund, et. al., 1965)

The disease was noted to occur most frequently in a severe form characterized by intermittent fever triggered by the appearance of trypanosomes in the blood. The appetite was unaffected, but in spite of this there was gradual emaciation. There was a copious, serous nasal discharge, patchy alopecia, petichiae and ecchymoses of all visible mucous membranes and a degree of incoordination. (Smith and Jones, 1957) Edematous swellings of the

lower limbs, abdomen and ventral thorax were often quite marked and proved a ready location from which to obtain material to visualize the etiological agent. (Siegmund, et. al., 1965) Icterus and progressive anemia eventually led to a fatal termination. (Smith, and Jones, 1957)

Diagnosis depended upon demonstration of the causal agent in blood smears or edematous fluid or by complement fixation if the infection was not patent. The complement fixation test did however, give both false positive and negative reactions. (Siegmund, et. al., 1965)

#### IV. Trypanosoma theileri

T. theileri is a large agent that occurs with world-wide distribution. It has been demonstrated in many hosts, the most important of which was the ox.

This agent is usually considered non-pathogenic. Under conditions of stress or intercurrent infection, however, the symptoms of acute trypanosomiasis and even death may develop quite rapidly. (Siegmund, et. al., 1965; Malmquist, 1965; Ewing and Carnahan, 1967)

Symptoms consisted of depression, progressive anemia, albuminuria and on occasion, death. (Siegmund, et. al., 1965)

Diagnosis of this condition relies on visualization and identification of the organism by techniques mentioned previously in this paper.

In addition to the diseases previously mentioned, the following conditions occur with unknown frequency in Western Africa



and probably in Nigeria. These conditions will be listed in numerical order under the general classification of the etiological agent.

#### Bacterial Infections

1. Brucellosis
2. Tuberculosis
3. Melioidosis
4. Actinomycosis
5. Spirochaetosis
6. Botulism

#### Protozoan Infections

1. Texas Fever
2. Eperythrozoonosis
3. Theileriosis
4. Babesiosis
5. Trichomoniasis

#### Viral and Rickettsial

1. African Horse Sickness
2. Rabies
3. Ephemeral Fever
4. Sweating Sickness
5. Infectious Bovine Infertility
6. Blue Tongue

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HISTOLOGICAL AND PATHOLOGICAL TECHNIQUES  
UTILIZED IN THE DIAGNOSIS OF CERTAIN  
ANIMAL DISEASES INDIGENOUS TO NIGERIA

by

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AN ABSTRACT OF A MASTER'S REPORT

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Nigeria, particularly the Northern Region, is basically an agricultural country, relying heavily on the livestock industry to assist in the support of the economy. The Nigerians, at the present time, are not able to realize all the benefits possible from this industry, due to the livestock losses which occur from disease processes, and also to the lack of facilities and trained personnel with which to stem a disease outbreak.

The diseases present in Nigeria are disseminated and control further hindered by certain husbandry practices such as the nomadic nature of some of the tribes, the Fulani herdsmen, and their cattle. Traveling about the country with their cattle they remain in one place only as long as the pasture remains good.

Diseases themselves have helped to produce conditions conducive to their spread as well as the spread of unrelated maladies. The tsetse fly, vector of trypanosomiasis, for instance, occupies a great portion of the Southern Region of Nigeria. It has been responsible for such a high trypanosomiasis incidence as to prevent large-scale cattle production without periodical anti-trypanosomal therapy. The Southern Region, therefore, depends quite substantially on cattle trekked from the north for their fresh meat supply.

This, largely unsupervised, movement of cattle gives any disease process an excellent opportunity to present itself.

With the foregoing facts in mind it is simple to understand how an easily performed early field diagnosis is imperative to control the spread of contagion in this West African country. There are many notorious animal diseases included in this report such as anthrax, contagious bovine pleural pneumonia, foot and mouth disease,

rinderpest and trypanosomiasis, and such little known diseases as cutaneous streptothricosis and heartwater. Any of these conditions enjoy the possibility of inflicting economical loss on the cattle population of any country, at any time.

It was the intent therefore, to report on certain animal diseases which have occurred naturally, primarily in cattle, and to review the general symptomatology, means of transmission, etiological agents, vectors where applicable and means of diagnosing these conditions with emphasis on field diagnosis. Clearly, if these conditions could be eradicated or at best controlled, the Nigerian economy would benefit greatly.

It was also the hope of the author that this report could provide an insight, however meager, into some of the disease problems, which may assist in the orientation of veterinarians and animal husbandrymen contemplating a tour of duty in this West African country.